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PHYTOPATHOLOGY

VOLUME XI NUMBER 11

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EFFECT OF FERTILIZERS AND LIME ON CONTROL OF TOMATO LEAF SPOT (SEPTORIA LYCOPERSICI)

F. J. Pritchard and W. S. Porte

With Sixteen Figures in the Text

INTRODUCTION

Observations and experiments covering a period of nearly 60 years seem to indicate that fertilizers and lime may be used to increase or decrease the amount of disease in a crop. The processes by which fertilizers bring about these changes, however, still need investigation. Sheldon (21, 22), Peltier (16), and Fromme and Murray (8) believe that treatments or conditions that cause a vigorous growth make the plants more susceptible to disease while those that delay or retard growth make them more resistant. Support of this view seems to be found, in part at least, in the results of Liebig (11), Roberts (18), Laurent (10), Marchal (14), Delacroix (3), McAlpine (12), Spinks (23), McCue and Pelton (13), Butler (2), and Thomas (25), who reported increased susceptibility to rust, mildew, and other foliage diseases from the use of nitrogenous fertilizers and manures. In most of these cases it was assumed that succulence of the tissues caused by nitrogen facilitated parasitic invasion.

Another current interpretation of fertilizer effects on disease resistance is that they modify the acidity of the soil and cell sap and thereby affect the activities of the parasites. In the case of soil inhabiting parasites this is quite obvious. The ginseng root-rot fungus, *Thielavia basicola*, as reported by Whetzel (30), is easily controlled by applying phosphate, especially acid phosphate to the ginseng beds. An acid soil also helps to control potato scab. On the other hand, the club root organism, *Plasmodiophora brassicae*, as reported by Stewart (24) and by Hendrick (9), may be largely controlled in cabbage and turnip fields by the use of lime.

That the acidity of the plant sap can be modified by the acidity of the soil has been reported on the basis of experiments, by Truog (27) and by Truog and Meacham (28). If these results should be substantiated by future investigations, it would seem that fertilizer ingredients that increase the acidity of the soil may also increase the acidity of the cell sap.

Delacroix (4) believes that phosphate inhibits disease by increasing the acidity of the cell sap, and Wagner (29) reports that the acidity (hydrogen-ion concentration) of plants injected with pathogenic bacteria rises simultaneously with the appearance of the first disease symptoms. These results would therefore seem to indicate that this is a means by which the plant protects itself against the action of parasites.

The use of phosphates for the control of plant diseases has given somewhat variable results. Laurent (10), Delacroix (4), Spinks (23), and McCue and Pelton (13) found that it increased resistance while Marchal (14), and Butler (2), and in one instance Laurent (10), found that it decreased it. These discrepancies may be due to peculiarities of the different organisms worked with or to variable effects of phosphates on different soils.

Lime has been reported by Ducomet (5) and by Delacroix (4), to decrease disease resistance, and by Marchal (14), Ramsbottom (17), and Thomas (25), to increase it. Notwithstanding the fact that Truog (27), Truog and Meacham (28), and Parker and Truog (15) have shown that it plays an important role in neutralizing and precipitating acids formed in protein metabolism, it is doubtful in view of these conflicting reports on disease control what influence, if any, it has on disease resistance.

The use of potash seems to have given more uniform results in the control of plant diseases than any other fertilizer ingredient. Marchal (14), Spinks (23), Russell (19), Finlow (6), and Butler (2), have published fairly reliable evidence on its effect in decreasing disease. Whether its action is due to the production of firmer tissues which tend to resist parasites or to an increased acidity of the cell sap and protoplasm has not been determined. Increased acidity of tomato fruits from the use of potash, as reported by Patterson (1) and by Tracy (26), would seem, however, to support the latter view.

INVESTIGATION

As tomato fields in the East and Middle West, where tomatoes are commonly grown for canneries, are badly infested by the tomato leaf spot fungus (*Septoria lycopersici*), the writers carried on numerous greenhouse experiments to determine whether certain quantities and ratios of fertilizers and lime may be used to help control this disease.

MATERIAL AND METHODS

The experiments were run in a number of different sets, each of which consisted of several fertilizer groups and a control group. Twenty-five plants were used in each group. One batch of medium fertile, friable, loam soil thoroughly mixed and sifted was used for all of the pot experiments.

Sodium nitrate (15.65% N), potassium sulphate (48% K_2O), and acid phosphate (16% available P_2O_5) were used in varying quantities both without other fertilizers and with a constant quantity of other fertilizer ingredients. All quantities of fertilizers used were calculated on the basis of parts per million of soil. Copper sulphate was also used in two sets of experiments.

The acid phosphate and lime were pulverized in a mortar and mixed with the sifted soil before it was potted: the sodium nitrate and potassium sulphate were dissolved in water and added to each pot by means of a pipette.

The plants receiving varying quantities of sodium nitrate were grown in jelly glasses; the others, in clay pots with closed bottoms.

All groups within a set were started on the same day by planting in the pots small tomato seedlings in the second leaf and keeping them moderately moist.

All the plants of each set were also inoculated with *Septoria lycopersici* at one time and kept in a moist chamber 48 to 60 hours and then transferred to benches in the greenhouse. One batch of spore material kept thoroughly mixed by frequent shaking was applied as uniformly as possible to all plants of a set by means of a compressed air sprayer. The initial infections were counted the eleventh day after inoculation.

The leaf areas and number of infections per plant and per unit area of leaf surface were recorded for both the treated plants and the controls.

The average leaf area per plant for each group of a set was determined by making leaf tracings of three average plants of each group and by measuring the tracings by means of a planimeter. One-third of the total leaf area of the three plants was used as the average leaf area per plant for the group.

RESULTS OF EXPERIMENTS

In figures 1 to 16 the controls are represented in the vertical columns marked zero. They received either no fertilizer as shown in figures 2, 4, 6, 8, 10, 12, 14, and 16 or uniform quantities of certain fertilizers used as constants for both the controls and treated plants, as noted in the headings of the other figures. The treated plants received nitrate, phosphate, or potash at the rates of 50, 150, 300, and 500 parts per million of soil, or lime or copper sulphate at the rates specified at the bottom of the graphs for these substances.

The graphs composed of broken lines connecting the dots show the relationship between the controls, which received no fertilizer variant, and the treated plants. Each dot expresses the average leaf area or number of infections per plant or per square inch of leaf surface for a

group of 25 plants. The continuous lines indicate the directions of the graphs.

Relationship between leaf area and number of infections per plant. The effect of nitrate, as shown in figures 1 and 2, was to increase both the leaf area and the number of infections per plant. Acid phosphate increased the leaf area and the number of infections per plant in three sets of experiments (Figs. 3, 5, 6) and decreased them in another (Fig. 4). Positive correlation between leaf area and number of infections per plant was also obtained for potash (Figs. 13, 14), for two sets of experiments with air-slaked lime (Figs. 7, 8) and for one with copper sulphate (Fig. 15). The exceptions in the few remaining sets of experiments are probably due to experimental error. These experiments therefore seem to indicate that fertilizers either augment or diminish the number of infections per plant by altering the leaf area.

Relationship between growth and susceptibility to infection. It might be assumed on the basis of the foregoing evidence that when the plants are uniformly inoculated and kept in an environment favorable for infection the number of infections per plant depends wholly on the amount of leaf area that may be infected were it not for the fact that a similar relationship seems to obtain between area of the leaves and number of infections per square inch of leaf surface. In figures 2, 3, 5, 7, and 14, for instance, leaf area and number of infections per unit area of leaf surface were increased while in figures 4, 8, 13, and 15 both were decreased.

TABLE 1

Effect of fertilizers and other substances applied to the soil on the development of tomato leaf spot.

TREATMENT	SQUARE INCHES OF LEAF AREA PER PLANT		INFECTIONS PER PLANT		INFECTIONS PER SQUARE INCH OF LEAF SURFACE	
	AVERAGE NUMBER	INCREASE IN PERCENTAGE	AVERAGE NUMBER	INCREASE IN PERCENTAGE	AVERAGE NUMBER	INCREASE IN PERCENTAGE
Nitrate	54.2	77	279	55	5.1	−13
Control	30.6		180		5.9	
Phosphate	81.8	14	341	50	4.1	32
Control	71.8		226		3.1	
Lime	63.2	−2	239	−8	3.7	−7
Control	64.4		261		4.0	
Potash	55.0	−19	309	−46	5.6	−33
Control	68.0		573		8.4	
Potash*	54.6	6	741	77	13.5	68
Control	51.6		417		8.0	
Copper Sulphate	100.0	−20	227	−17	2.3	4
Control	125.6		275		2.2	

* As the increased number of infections in this set of experiments is out of all proportion to the increase in leaf area this set was not averaged with the preceding one.

The relationship between leaf area and number of infections per plant and per unit area of leaf surface may also be seen in the summary of the results in table 1 in which is recorded the average of all groups of plants receiving the same fertilizer or other variant and the average of the controls for these groups.

The correlation between growth, as measured by leaf area, and number of infections per plant is clearly brought out in the above table. The correlation between leaf area and number of infections per unit area of leaf surface is also obvious although not so well supported in the summary of the nitrate and copper sulphate treatments as in the separate graphs. When the impossibility of inoculating all groups of plants uniformly is considered, it is not surprising that there are some exceptions. It would therefore seem that conditions favorable for growth not only increase the leaf area and thereby produce more surface for infection but also increase the susceptibility of the plants to disease, while conditions that hinder growth reduce susceptibility.

DISCUSSION

No fertilizer or other substance used seemed to increase or decrease the susceptibility of the tomato plants to leaf spot except as it affected growth or the internal conditions accompanying growth. This was true also of the different ratios and quantities of fertilizers used.

As the plants used in the foregoing experiments were heavily inoculated and kept under ideal conditions for infection, the number of initial infections is much higher than would ordinarily occur naturally in the field in the early part of the growing season. The contrast would also be more marked in the secondary infections as the more heavily infected plants would produce the more spores. Moreover, in the field the larger and heavier plants would hold rain and dew longer and therefore afford better opportunities for infection. It is therefore obvious that excessive vegetative growth should be avoided.

It is somewhat difficult to compare the results of these experiments with those previously published as most previous experiments of this kind were made on a different basis and the results were expressed either as general conclusions or as percentages of infection. Although Thomas (25) has published his results in the form of numbers of infections per plant or per leaf he has published no data on number of infections per unit area of leaf surface. In fact some of these previous reports contain only observations made on plats or fields in which different fertilizers were used primarily for another purpose. The data, as might be expected, under such circumstances, is therefore scanty. Not only were the crops and parasites different but the infections were often of such a

spreading nature that a single infection of a leaf was ultimately equivalent to several infections. However, the interpretation of the effects of fertilizers in the present experiments would explain nearly all results previously published and also account for most of their apparent discrepancies.

As it has been found by Truog and Meacham (28), that high acidity (hydrogen-ion concentration) of the cell sap frequently restricts the growth of plants while a lower acidity favors growth, it might be assumed that the relationship between growth and infection is really a relationship between acidity and resistance. But as Haas (8) and others have shown that the total acidity (undissociated and dissociated) of plant cells is usually much greater than the dissociated acidity (hydrogen-ion concentration), it would seem that the neutralization of acid in the cell sap by lime or other substances would have very little effect on the hydrogen-ion concentration, for as soon as some of the free acid were precipitated more would immediately become dissociated. Nevertheless if dissociation of acids in plant cells does not take place so rapidly as assumed, hydrogen-ion concentration may have an important bearing on susceptibility to disease. It is doubtful, however, whether it is the plant's sole means of resisting parasites although it may be comparable to the serum of the blood in inhibiting animal diseases.

Although fertilizers apparently affect susceptibility of plants to disease and may be so used as to avoid conditions favorable for excessive infection, they do not seem to provide a sufficient means for the control of tomato leaf spot.

SUMMARY

Different quantities and ratios of sodium nitrate, potassium sulphate, acid phosphate, air-slaked lime, and copper sulphate were used in pots of soil to determine their effect on the control of tomato leaf spot. Treatments with these substances either increased or decreased the leaf area and thereby caused a corresponding alteration in the number of infections per plant. These alterations in leaf area were also correlated with similar alterations in number of infections per unit area of leaf surface. Favorable conditions for growth therefore increased susceptibility to infection while unfavorable conditions decreased it.

The relationship between plant growth and acidity of the cell sap reported by Truog (27, 28) and his associates may have an important bearing on susceptibility to infection, for number of infections per unit area of leaf surface is positively correlated with growth and growth, as shown by Truog (27) is, at least in some cases, inversely correlated with the acidity of the cell sap.

LITERATURE CITED

(1) BISHOP, W. H. and H. J. PATTERSON. Tomatoes. Maryland Agric. Exp. Sta. Bull. **11**: 47–73. 1890.

(2) BUTLER, E. J. Immunity and disease in plants. Agri. Journ. India, Spec. Indian Sci. Cong. Number **1918**: 1028. 1918.

(3) DELACROIX, GEORGES. Observations et recherches au sujet de la pourriture grise produite par le *Botrytis cinerea*. Bull. Mens. Off. Renseig. Agri. [Paris] **4**: 406–409. 1905.

(4) ——— Maladies des plantes cultivées—Maladies nonparasitaires. **1**, *431 p., 58 pl.* Paris. 1908.
Index bibliographique, p. 418–420.

(5) DUCOMET, VITAL. Pathologie végétale. Maladies parasitaires: champignons—bactéries. *x, 298 p., 21 fig.* 1908.

(6) FINLOW, R. S. Rhizoctonia in jute: The inhibiting effect of potash manuring. Agri. Jour. India, Spec. Indian Sci. Cong. Number **1918**: 65–72. 1918.

(8) FROMME, F. D. and T. J. MURRAY. Angular leafspot of tobacco, an underscribed bacterial disease. Jour. Agri. Res. **16**: 219–228. *Pl. 25–27.* 1919.
Literature cited, p. 227–228.

(8) HAAS, A. R. The reaction of plant protoplasm. Bot. Gaz. **63**: 232–235. 1917.

(9) HENDRICK, JAMES. The use of lime in controlling finger-and-toe in turnips. Trans. Highland and Agric. Soc. Scotland **30**: 137–145. 1918.

(10) LAURENT, EMILE. Recherches expérimentales sur les maladies des plantes. Ann. Inst. Pasteur **13**: 1–48. 1899.

(11) LIEBIG, JUSTUS VON. Einleitende worte Sitzungsber. Königl. Bayer. Akad. Wiss. München **1863** (Bd. 2): 375–380. 1863.

(12) MCALPINE, D. Handbook of fungus diseases of the potato in Australia and their treatment. *iii, 215 p., 50 pl. (pl. 1–2 col.).* Melbourne. 1911.

(13) MCCUE, C. A. and W. C. PELTON. Tomatoes for the canning factory. Delaware Agr. Exp. Sta. Bull. 101. *86 p., 2 fig., 2 pl.* 1913.

(14) MARCHAL, EMILE. De l'immunisation de la laitue contre le meunier. Compt. Rend. Acad. Sci. [Paris] **135**: 1067–1068. 1902.

(15) PARKER, F. W. and E. TRUOG. The relation between the calcium and the nitrogen content of plants and the function of calcium. Soil Sci. **10**: 49–56. *1 fig.* 1920.
References, p. 56.

(16) PELTIER, G. L. Susceptibility and resistance to citrus-canker of the wild relatives, citrus fruits, and hybrids of the genus Citrus. Jour. Agric. Res. **14**: 337–355. *Pl. 50–53.* 1918.
Literature cited, p. 356–357.

(17) RAMSBOTTOM, J. K. Iris leaf-blotch disease (*Heterosporium gracile* Sacc.). Jour. Roy. Hortic. Soc. **40**: 481–492. 1915.
Bibliography, p. 492.

(18) ROBERTS, I. P. The fertility of the land. *xvii, 415 p., 45 fig.* New York, London. 1897.

(19) RUSSEL, E. J. Soil conditions and plant growth. *viii, 168 p., 7 fig.* London, New York [etc.] 1912.
A selected bibliography, p. 154–166.

(20) ——— A student's book on soils and manures. *ix, 206 p., 34 fig.* Cambridge, (Eng.). 1915.
Bibliography, p. 202.

(21) SHELDON, J. L. Preliminary studies on the rusts of the asparagus and the carnation: parasitism of Darluca. Science, n. s. **16**: 235–237. 1902.

(22)——— The effect of different soils on the development of the carnation rust. Bot. Gaz. **40**: 225–229. 1905.

(23) SPINKS, G. T. Factors affecting susceptibility to disease in plants. Pt. 1. Jour. Agric. Sci. **5**: 231–247. *Pl. vii.* 1913.

(24) STEWART, F. C. Prevention of cabbage club-root. New York State Agric. Exp. Sta. Ann. Rept. **14** (1895): 525–529. 1896.

(25) THOMAS, H. E. The relation of the health of the host and other factors to infection of *Apium graveolens* by *Septoria apii*. Bull. Torrey Bot. Club **48**: 1–29. 1921.
Literature cited. p. 27–29.

(26) TRACY, W. W. Tomato culture. *x, 150 p., 43 fig.* New York. 1907.

(27) TRUOG, EMIL. Soil acidity: I. Its relation to the growth of plants. Soil Sci. **5**: 169–195. 1918.
References, p. 193–195.

(28)——— and M. R. MEACHAM. Soil acidity: II. Its relation to the acidity of the plant juice. Soil Sci. **7**: 469–474. 1919.
References, p. 474.

(29) WAGNER, R. J. Wasserstoffionenconzentration und natürliche Immunität der Pflanzen. Centralb. Bakt. Parasitenk. u. Infektionskrankh. **44**: 708–719. 1916.

(30) WHETZEL, H. H., J. ROSENBAUM, J. W. BRANN, and J. A. MCCLINTOCK. Ginseng diseases and their control. United States Dept. Agric. Farmers' Bull. 736. *23 p., 23 fig.* 1916.

DESCRIPTION OF FIGURES

FIG. 1. Effect of increased quantities of sodium nitrate and constant quantities of acid phosphate and potassium sulphate.

FIG. 2. Effect of varying amounts of sodium nitrate in the absence of other fertilizers.

FIG. 3. Effect of increased quantities of acid phosphate and constant quantities of sodium nitrate and potassium sulphate.

FIG. 4. Effect of varying amounts of acid phosphate in the absence of other fertilizers.

FIG. 5. Effect of increased quantities of acid phosphate and constant quantities of sodium nitrate and potassium sulphate.

FIG. 6. Effect of varying amounts of acid phosphate in the absence of other fertilizers.

FIG. 7. Effect of increased quantities of airslaked lime and constant quantities of acid phosphate, sodium nitrate, and potassium sulphate.

FIG. 8. Effect of varying amounts of air-slaked lime in the absence of fertilizers.

FIG. 9. Effect of increased quantities of air-slaked lime and constant quantities of acid phosphate, potassium sulphate, and sodium nitrate.

FIG. 10. Effect of varying amounts of air-slaked lime in the absence of fertilizers.

FIG. 11. Effect of increased quantities of air-slaked lime and constant quantities of acid phosphate, potassium sulphate, and sodium nitrate.

FIG. 12. Effect of varying amounts of air-slaked lime in the absence of fertilizers.

FIG. 13. Effect of increased quantities of potassium sulphate and constant quantities of acid phosphate and sodium nitrate.

FIG. 14. Effect of varying amounts of potassium sulphate in the absence of other fertilizers.

FIG. 15. Effect of increased quantities of copper sulphate and constant quantities of acid phosphate, potassium sulphate, and sodium nitrate.

FIG. 16. Effect of varying amounts of copper sulphate in the absence of fertilizers.

FIG 1 NITRATE VARIANT

AP 100 ppm.
K_2SO_4 100 ppm.

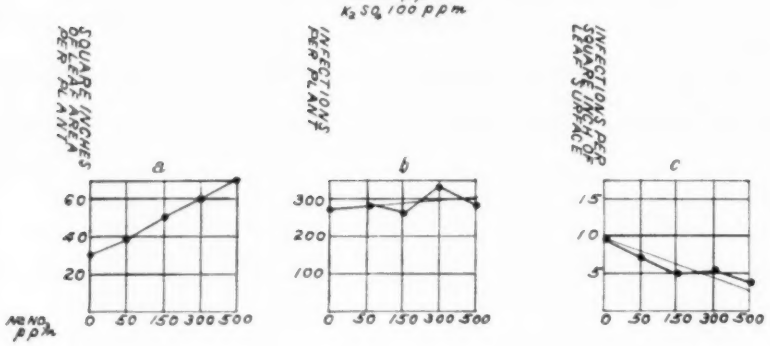


FIG 2 NITRATE VARIANT

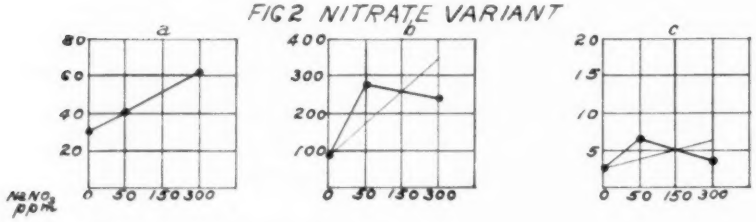
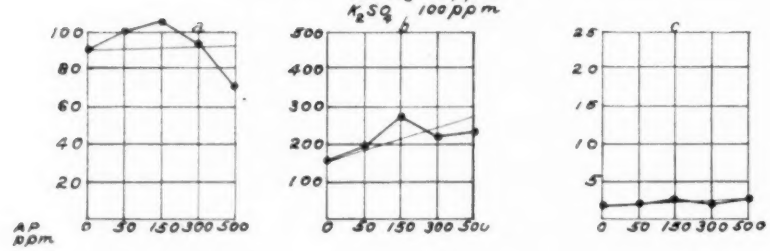


FIG 3 PHOSPHATE VARIANT

$NaNO_3$ 100 ppm.
K_2SO_4 100 ppm.



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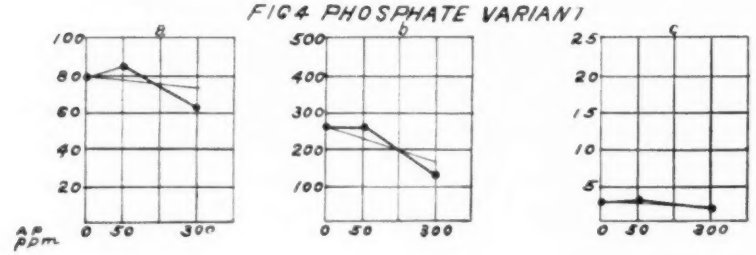


FIG 5 PHOSPHATE VARIANT

$NaNO_3$ 50 ppm
K_2SO_4 50 ppm

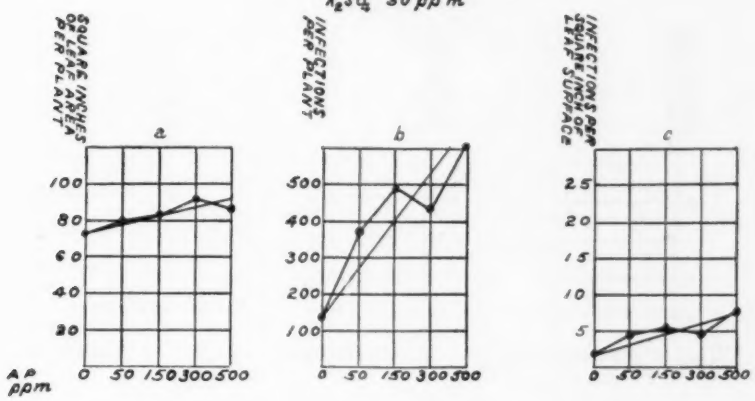


FIG 6 PHOSPHATE VARIANT

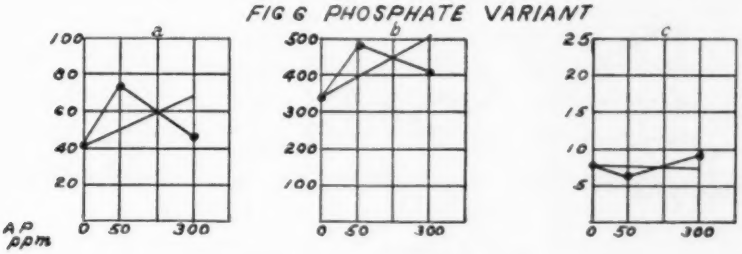


FIG 7 LIME VARIANT

A P 100 ppm
$NaNO_3$ 100 ppm
K_2SO_4 100 ppm

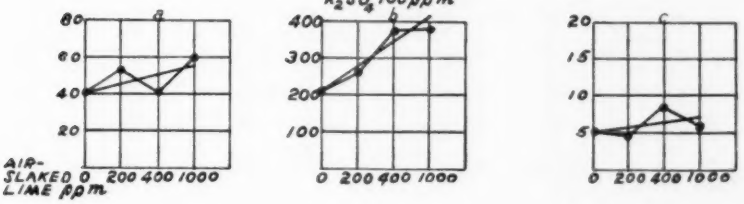


FIG 8 LIME VARIANT

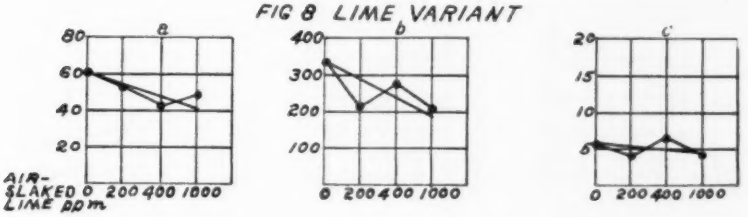


FIG 9 LIME VARIANT

AP 100 ppm
K_2SO_4 100 ppm
$NaNO_3$ 100 ppm

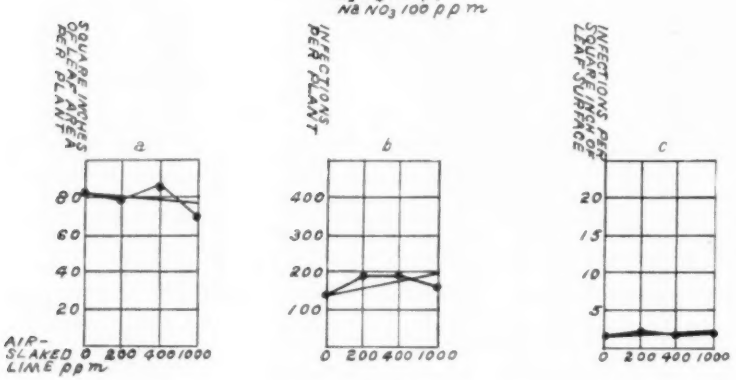


FIG 10 LIME VARIANT

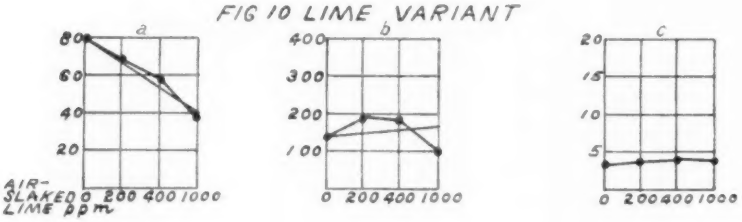


FIG 11 LIME VARIANT

AP 100 ppm
K_2SO_4 100 ppm
$NaNO_3$ 100 ppm

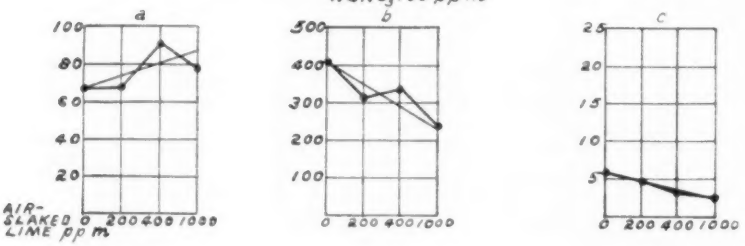


FIG 12 LIME VARIANT

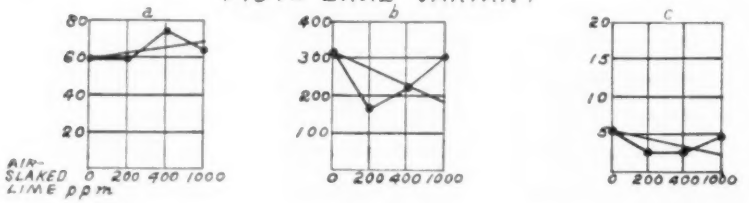


FIG 13 POTASH VARIANT
AP 100 ppm
$NaNO_3$ 100 ppm

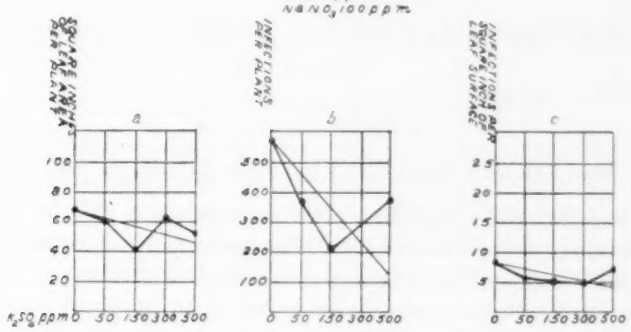


FIG 14 POTASH VARIANT

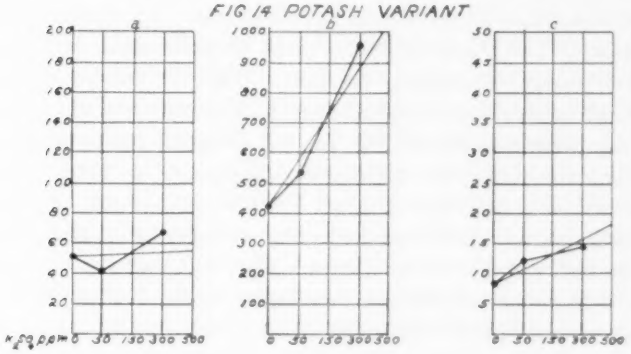


FIG 15 COPPER SULPHATE VARIANT
AP 100 ppm
K_2SO_4 100 ppm
$NaNO_3$ 100 ppm

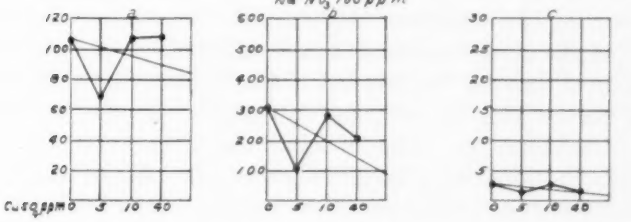
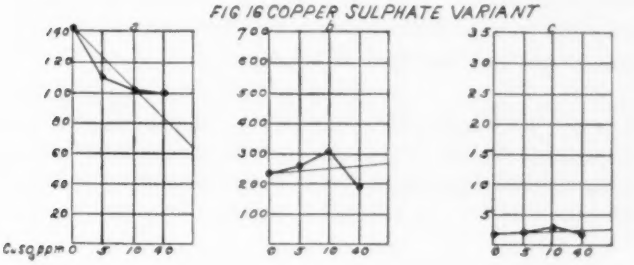


FIG 16 COPPER SULPHATE VARIANT



THE RELATION OF AIR TEMPERATURE TO CERTAIN PLANT DISEASES

JAMES JOHNSON[1]

WITH PLATES XXI TO XXIII AND TWO FIGURES IN THE TEXT

That atmospheric conditions play an important rôle in determining the occurrence and severity of plant diseases is too well known to warrant introductory remarks in a brief paper. It is a striking fact, however, that a decided difference of opinion exists as to the most favorable conditions for the development of many of our best known diseases. This is due in large part to published opinions based on more or less limited observation. The need for more exact experimental data on this subject is becoming increasingly evident. The difficulties encountered in attempts at controlling air conditions in the presence of sunlight are, of course, the main reasons for the limited progress made in this direction.

The need for controlled air conditions became so great in connection with the writer's problems that in 1919 he undertook the construction of air-conditioning apparatus without, perhaps, fully understanding the inevitable barriers which lay ahead. By "cut and try" methods, however, an apparatus was finally developed in the fall of 1920 which has given controlled conditions quite satisfactory for present needs. It is not the aim of the present paper to describe this apparatus in detail, or to present a finished piece of research conducted with it, but rather to indicate the needs and possibilities for similar lines of work. A brief description of the chambers is essential, however, in order that the method may be understood and the reliability of the results obtained may be judged. Primarily for purposes of illustration three distinct types of diseases have been given most attention. These are: the mosaic disease of tobacco, the cause of which is as yet uncertain: the "wildfire" disease of tobacco, a bacterial leaf spot caused by *B. tabacum* Wolf and Foster; and the late blight of the potato, a fungus disease caused by *Phytophthora infestans*. Several other diseases have also been worked with, including tomato wilt caused by *B. solanaceranum* Erw. F. S. and an unnamed bacterial leaf spot of tobacco.

[1] Coöperative investigations of the Office of Tobacco Investigations, Bureau of Plant Industry, United States Department of Agriculture, and the Wisconsin Agricultural Experiment Station. Published with the permission of the Director of the Wisconsin Agricultural Experiment Station and the Secretary of Agriculture of the United States.

It is not proposed to review the literature bearing upon the subject of atmospheric conditions in relation to plant diseases, since this would be too voluminous. Brief reference will be made only to certain papers bearing directly on the subject.

DESCRIPTION OF AIR-CONTROL CHAMBERS

The experiments to be described were conducted in three simultaneously operated chambers located in a greenhouse unit at the Wisconsin Agricultural Experiment Station. This greenhouse was heated with steam and fitted with thermostatic control and while ordinarily the temperature variation was about 5°, the range at times ran as high as 15° F.

The chambers fitted with air-control are composed essentially of five parts: the chambers proper, with due consideration for adequate insulation and light; an electric heater system in each chamber under thermostatic control; refrigeration within the cooler chambers; an automatically controlled humidifying system for each chamber; and means for renewal and circulation of air within the chambers.

Construction of chambers. The chambers are four foot cubes with three sides and bottom of wood and the top and one side of glass (Pl. XXI, fig. 1). Illumination was sacrificed in order to control more readily the air condition; but, as will be shown, the plants did not suffer esriously from lack of light during the period of exposure in the chambers, which was usually two weeks. Experience has shown that it is practically impossible to control the temperature in chambers constructed largely of glass exposed to full sunlight.

The bottom and three sides are of double walled ¾ inch cypress, with building paper and a dead air space between. The top and one side (the doors) are of two layers of single strength glass with air space between. The sides closed with glass also have wooden doors which can be closed for better insulation when light is not desired for certain types of experiments.

Heating system. The chambers are heated with 250 or 500 Watt Westinghouse heating units, regulated with Ostwald's toluene-mercury thermostats. The break of the heating circuit is therefore made by a relay operated through the thermostat by means of a direct current of two or three volts from storage batteries or from dry cells. The special feature introduced here is a double control system, that is, two thermostats, two relays (Pl. XXI, fig. 3) (connected in series) and two separate batteries for each chamber so that in case of accidental failure on the part of either the primary thermostat, relay or battery to operate, the secondary set would come into play following a small rise in temperature

This system obviated danger from overheating due to failure of apparatus to operate. A drop in temperature could result only from blowing out of fuses or burning out of the heating units.

Refrigeration. Artificial refrigeration was not available, and consequently ice had to be used. A small brine system was installed for one of the chambers, the primary coil (¾ inch) being in an ice chest holding about 500 pounds of crushed ice and the secondary coil being distributed over three inside walls of the chamber. Calcium chloride brine was circulated through the coils by means of a small rotary pump operated by a one-half horse power motor (Pl. XXI, fig. 2). The circulation of the brine was controlled by fitting the motor with thermostatic control. This gave fairly good results, although the variation in temperature was somewhat greater than in the heated chambers due to the "lag" of the cooling. The efficiency of the system was not more than 8–10° C below that of the greenhouse temperature, however, so that temperatures as low as 12 or 15° C could be maintained only during the winter months when the greenhouse temperature could be kept at or below 22° C.

In case of failure on the part of the brine system to operate, or when it was desired to save ice, a secondary cooling device could be brought into operation provided sufficiently cool weather existed out of doors. This consisted simply in blowing air from outside the greenhouse into the chamber by means of a blower (Pl. XXI, fig. 2, E) with thermostatic control. Excessive lowering of the temperature from refrigeration was guarded against by heaters connected with thermostats. It was also found necessary to introduce a small "check chamber" to prevent flow of cold air into the experimental chamber.

Humidification. The source of humidity consisted essentially of a current of air passing through a spray of water held at a temperature a few degrees above that of the chamber to be humidified. The water supply for all the chambers was heated by steam in a 40-gallon hot-water-tank fitted with a thermostat (Pl. XXI, fig. 4). By mixing the heated and cold water in a small "mixer" near the spray the proper temperature (as read by mercury thermometers set in oil wells in the mixers) of spray water could be secured for each chamber. The water passed through two screens before passing out through an ordinary mist type fruit tree spray nozzle. The spray was confined in galvanized iron tanks 5 feet high and 15 inches in diameter (Pl. XXI, fig. 3, J), with a drain at the base running into the sewer. Number O "Sirroco" blowers (1/20 H. P.) were used to blow air in at the base through the spray and out through a three inch pipe near the top, which led the current of warmed and saturated air into the chamber. The 110 volt current operating the blowers was connected through a relay, operated by direct current from

batteries through a "humidostat" in the chambers. This "humidostat" was simply a hygrograph with a platinum wire soldered to the pen arm so as to dip into a cup of mercury. When the relay circuit is completed through lowering of humidity, and resultant lowering of the wire into the mercury cup, the blower is operated until sufficient moist air is blown into the chamber to raise the pen arm and break the motor current circuit. At the same time the hygrograph pen draws the curve giving the humidity record. With proper adjustment quite constant humidity could be maintained in this way, the variation normally not being more than two or three percent (Fig. 1). This method can be worked satisfactorily only for high humidities. For running lower humidities it is planned to reverse the system; that is, pass the air through a spray of water colder than the chamber, thus reducing its moisture holding capacity before passing into the chambers. This method has not been tested sufficiently as yet to warrant statements in regard to its successful operation.[1]

Renewal and circulation of air. The renewal of the air is accomplished through the humidifying apparatus. Normally, fresh air is blown into the chamber every five or ten minutes. The circulation of the air within the chambers was accomplished by means of 13 inch fans running continuously in each chamber at a speed which kept the air in fairly rapid motion and absolutely prevented stratification. The fans were all operated by one motor (1/6 H. P.) (Pl. XXI, fig. 2, F) belted to the fan shafts. The speed of all the fans was therefore the same.

Operation of the chambers. Low growing plants were usually set on small tables in the chambers so as to bring them near the upper glass. Opening the doors to water the plants resulted in a rapid drop of humidity and of temperature which, though these were raised with almost equal rapidity when the doors were closed, usually resulted in disfigured curves. The plants therefore were set in large pans into which water was poured through a hose connected with a funnel on the outside, so that the plants were subirrigated, making it unnecessary ordinarily to open the doors more than once a week in order that the records might be changed.

The hygrographs and thermographs were relied upon only to show variation. The actual temperature readings were taken daily, and the humidity readings occasionally by means of a psychrometer set in front of a small electric fan which served to blow a strong current of air on the thermometers.

The evaporating power of the air was different, of course, at the different temperatures, even though the relative humidity might be the same in all cases. Three Livingston atmometers were used in each

[1] Since this paper was written a humidostat manufactured by the Johnson Service Company of Milwaukee has been used with good results.

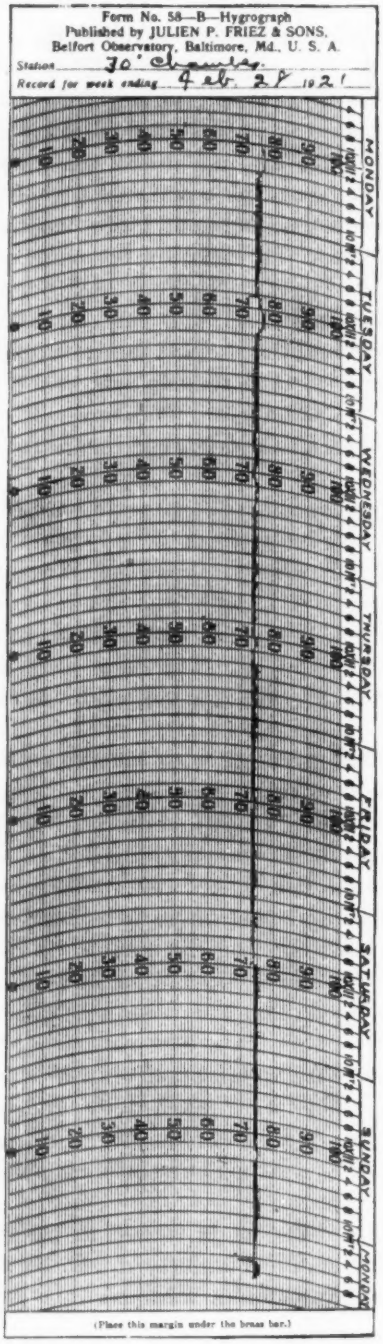


FIG. 1. HYGROGRAPH RECORD ILLUSTRATING THE DEGREE OF HUMIDITY CONTROL OBTAINED WITH GROWING PLANTS.

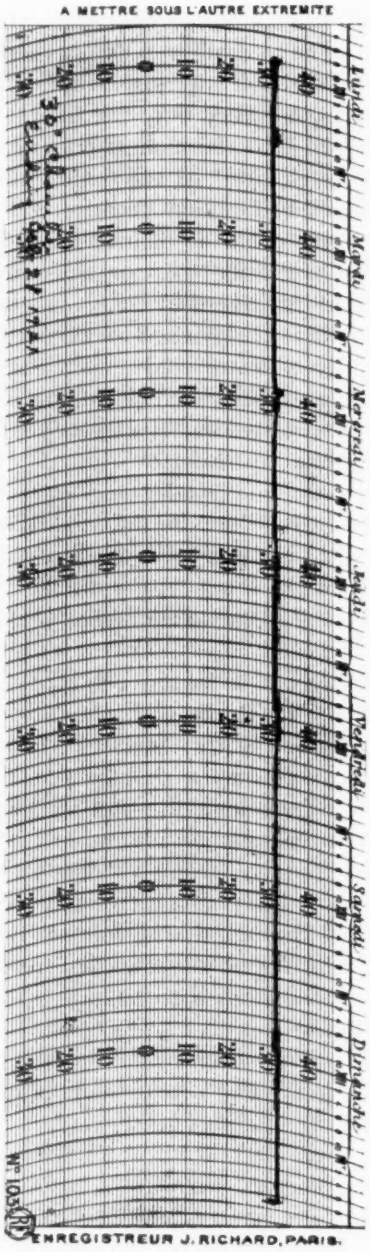


FIG. 2. THERMOGRAPH RECORD ILLUSTRATING THE TEMPERATURE CONTROL OBTAINED WITH GROWING PLANTS.

chamber to measure this factor. Time was not taken to secure adjustment for obtaining the same evaporating power in each chamber, but this was approximated in some instances, and was determined in all cases, so that its influence could be considered.

Two principal difficulties arise in operating the chambers. The first of these is condensation of moisture on the glass at high temperatures and high humidity. The second is the disturbing action of strong sunlight on the temperature of the chambers beginning with the spring months. Shading during part of the day was, therefore, necessary at times. These difficulties are not regarded, however, as having influenced the final results obtained in the experiments.

Since these chambers were completed the writer has had the opportunity of hearing discussions by Dr. Hottes of the University of Illinois on temperature and humidity control. A brief note has been published on his chambers (3). Parties interested in temperature and humidity apparatus construction should not fail to take advantage of Dr. Hottes' valuable ideas and long experience in this line before undertaking new construction.

RELATION OF TEMPERATURE TO MOSAIC

Very little experimental data and only very limited observations exist with regard to the influence of temperature on mosaic. Doolittle (2) recently published a brief statement on the cucumber mosaic to the effect that . . . . "at soil temperatures between 22° and 27° C., the percentage of infection was the same, although the incubation period increased with lower temperatures. Increasing the soil temperature from 27° to 30° C., regardless of air temperature, reduced the incubation period from six to three days and produced a higher percentage of infection, accompanied by symptoms of a new type."

It is quite apparent that the development of mosaic symptoms is closely correlated with rate of growth in infected plants. Since soil temperature affects the rate of growth it is to be expected that it will also affect the incubation period for mosaic. The writer has undertaken, however, to study the effect of temperature upon the mosaic virus, itself, as measured by its rate of activity in the living plant, although to be sure we still cannot separate it completely from growth responses in the plant.

In studying the influence of temperature on the tobacco mosaic two methods have been used. In the first, the plants were inoculated and immediately placed in the chambers, where they were left for fourteen days, after which all were taken out and set on a greenhouse bench at one series of temperatures (usually a daily variation from 18–24° C.) where the progress of the disease was further observed. The second method consisted in placing plants already showing striking symptoms of the

disease in the chambers and observing its further development. The incubation period was longer in the short, dark days of the winter months than in February and March, owing apparently to a lower rate of growth. The results of six experiments are shown in table 1.

The data show a marked influence of air temperature on the incubation period for mosaic in tobacco. The absolute minimum possibly can never be determined since some host development must apparently occur in order to obtain the symptoms, and hence the incubation period could be theoretically lengthened indefinitely by holding the host at a temperature at which no growth is made. From this and other data the optimal temperature for the activity of the virus appears to be between 28 and 30° C and the maximal temperature is close to 36° C (Pl. XXI, fig. 1).

A significant condition arises when plants with all leaves already badly affected are placed at a temperature of 36–37° for about two weeks. The new leaves which develop are free from mosaic symptoms and most of the older leaves which are mottled but still in a growing condition gradually lose their mottled appearance and become apparently normal as far as symptoms go. This is due probably to the retarding of the deleterious action on chlorophyll production, nevertheless the virus is still present as shown by the reappearance of the symptoms when the plants are again subjected to lower temperatures. Apparently a similiar "recovery" of new leaves may be obtained by forcing a mosaic plant to grow by means of a favorable soil temperature in an air temperature considerably below the optimum for the virus, or by exposure of a mosaic plant to a constant temperature too low for good development of the disease but permitting some plant growth.

The experiments with mosaic were undertaken with the hope that some indirect evidence as to the nature of the casual agent might be established. Further experiments are planned to support the evidence secured in this direction. The general trend of the evidence however may be presented here. The correlation between the response of the mosaic virus with respect to temperature and that of living organisms is evident from table 1. Enzymatic action varies with temperature in a similiar manner, but the literature is uniformally agreed that the optimal temperature for enzymatic activity lies usually between 37° and 40° C, with the added assurance that their activity in the presence of their normal substrata is even higher. The influence of length of exposure to the temperature in question becomes an important factor to consider in this connection, however, and the effect of long exposure to comparatively low temperatures is as yet not adequately understood. If we assume that plant parasites are tissue destroyers through the activity of their respective enzymes we have evidence in these experiments that the

TABLE 1

Influence of air temperature on mosaic of tobacco

EXPERIMENT NUMBER AND DATE	TEMPERATURE C°	RELATIVE HUMIDITY C. C.	AVERAGE DAILY EVAPORATION	NUMBER OF PLANTS INOCULATED	NUMBER OF PLANTS INFECTED AFTER (DAYS)								RELATIVE GROWTH AFTER 14 DAYS		
					IN CHAMBERS				OUTSIDE CHAMBERS				AVERAGE NO. NEW LEAVES	AVERAGE INC. LGTH. LEAVES (CM.)	AVERAGE INCREASE OF HEIGHT (CM.)
					8	10	12	14	18	22	26	30			
5	12–15	69–81	8.1	5				0	0		0	5			
1–17–30	25–26	89–92	9.2	5				1	5		5	5			
	34–35	82–86	17.1	5				0	0		4	5			
6	13–15	60–90	9.2	7				0	0	0	0	4			
2–1–13	25–26	79–83	9.4	7				5	7	7	7	7			
	34–35	82–86	17.8	7				0	0	5	7	7			
7*	17–21	80–85	7.3	9	0	0	0	0	5	9	9	9	1.2	28.5	1.0
2–14–28	30–31	78–80	14.1	10	1	10	10	10	10	10	10	10	2.5	38.8	8.8
	35–37	82–89	14.9	9	0	0	0	0	0	0	3	5	1.7	34.4	10.0
8	27–29	80–85	12.4	10		7	8	9	10				3.6	74.7	7.7
2–28–3–14	30–31	77–82	19.2	10		8	9	10	10				3.2	61.9	10.1
	33–34	82–89	13.3	10		1	6	8	10				3.4	53.6	6.6
9	28–30	80–85	13.0	9	4	9	9	9	9	9	9	9	4.0	66.3	8.2
3–14–28	31–32	81–83	19.3	9	1	5	7	7	9	9	9	9	3.6	55.1	6.9
	35–37	82–85	14.4	9	0	3	3	3†	3	5	6	7	3.6	48.9	6.4
10	18–22	70–80	12.5	10	0	0	0	0	7	9	10	10	3.5	56.2	4.0
3–28–4–10	29–30	88–90	7.8	10	9	10	10	10	10	10	10	10	4.8	78.5	11.8
	36–38	80–86	14.5	10	0	0	0	0	0	7	9	10	4.0	50.9	6.2

* Fuse blown out and temperature dropped proportionally in all chambers for about 12 hours on Feb. 17.

† Symptoms questionable and visible only by transmitted light.

enzyme or enzymes of *B. solanaceraum*, for instance, is at its height of activity at a temperature where the mosaic virus is no longer active (Pl. XXIII, fig. 2). Again, the metabolic processes in growing plants are now regarded as being dependent in a large part upon the activity of enzymes, and as may be seen from table 1, the growth activity of plants subjected to a temperature of 34–37° C. was not seriously affected as compared with the effect upon the mosaic virus.

Referring more specifically to the enzymes which are said by the supporters of the enzyme theory (1, 6) to be responsible for the mosaic disease of tobacco, namely, the oxidases principally, and, perhaps, the peroxidases and catalases, we can find only contradictory evidence in the temperature studies. If mosaic is due to the increased activity of these enzymes we should find them in greatest abundance at a temperature of 28–30° (in mosaic plants) but with their activity greatly reduced if not entirely eliminated in plants exposed for some time at a temperature of 36–37,° where the mosaic virus is no longer active as shown by the "recovery" of mosaic plants.

Qualitative and quantitative determinations of these enzymes do not indicate such reduced activity at the higher temperatures and consequently the part played by these enzymes as a causal agent in the mosaic disease is open to serious question. The increased activity of the oxidases in mosaic leaves seems to be therefore a result rather than a cause of the disease.

It is not to be expected that soil temperature in itself can directly affect the activity of the virus of mosaic. Since the appearance of mosaic symptoms is dependent very largely upon the rate of growth of the host, however, it follows that correlation between soil temperature and the mosaic symptoms will exist. Similarly, air temperature also must affect the incubation period for mosaic since it also influences the rate of growth of the host. With air temperature we have however the additional affect of temperature upon the activity of the virus. The determination of the relative importance of temperature on three different factors as influencing the symptoms of mosaic becomes very complex, particularly since the expression of each of these factors is dependent one upon the other.

Two experiments have been conducted to determine the influence of soil temperature upon the mosaic disease of tobacco. The first experiment was carried on in November and December 1920, with soil temperatures ranging from 15 to 35° C. Inoculations were made at three different times in the course of about six weeks with no symptoms occurring at any temperature, excepting finally at a soil temperature of 35° C. The plants at 25 and 30° became "frenched," however, which may have partially obscured the mosaic symptoms. The light condi-

tions were poor and the plants did not grow rapidly although they did increase their size two to four fold. The greenhouse temperature was purposely held low (below 20° C) to reduce the refrigeration necessary for the chambers with air control which were in the same house. It is now believed that the long incubation period in this experiment was largely due to the low air temperature prevailing.

In March 1921 another series of soil temperature tests was started under good light conditions and a relatively high air temperature (23 – 28°). The first symptoms were evidenced in 7 days at a soil temperature of 27–28,° followed on the eighth day by symptoms at 23–24° and at 30–32.° C. On the ninth day 100 per cent infection was found at soil temperatures of 27–28,° 31–32,° 34–36,°with 50 per cent at 23–24° and none at 19–20.° It seems reasonable to assume from these data and from the results in the chambers that the influence of soil temperature upon mosaic is largely a question of growth response in the host plant, that air temperature is of primary signficance, and that a soil temperature unfavorable for host development will increase the incubation period, even when an air temperature favorable for the host plant and the virus is maintained.

THE "WILD-FIRE" LEAF-SPOT OF TOBACCO

Tobacco plants inoculated with the "wildfire" organism *Bacterium tabacum* Wolf and Foster were placed in the chambers simultaneously with the mosaic disease series. Infection was produced through needle pricks from a suspension of the organism in water. Infection without wounding has not proved uniform and reliable, consequently the above method was used. Three or four leaves on each plant were inoculated with 10 or 12 punctures on each leaf. The relative results are not readily expressed in per cent of infection (which was always 100 per cent unless below the minimum or above the maximum) or in size of lesions since the "halo" areas grade rather indefinitely into healthy areas. The data are therefore merely expressed in terms of relative amount of infection based on rapidity of infection and estimation of size of chlorotic ("halo") areas.

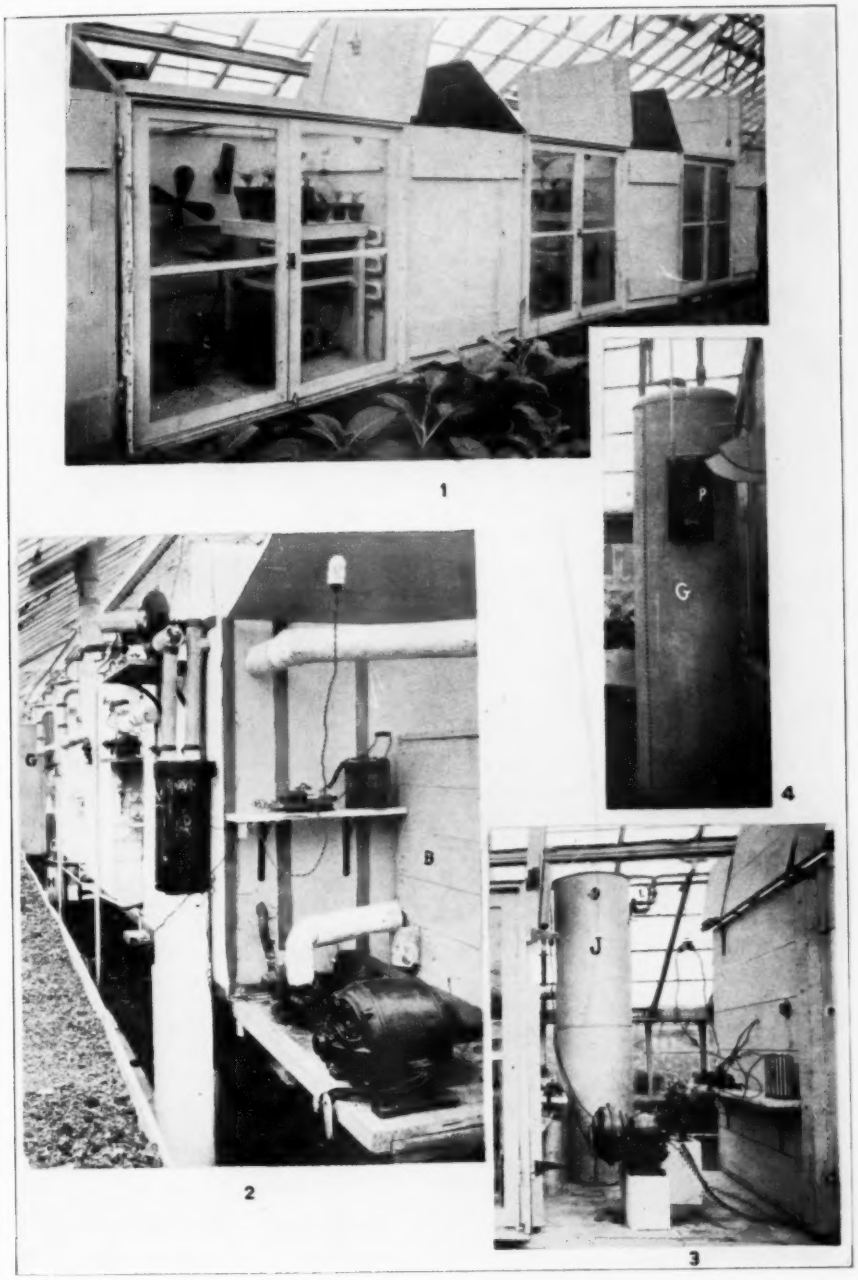
The evidence is quite conclusive that at the humidities prevailing and in wounded leaves the progress of the wildfire disease is very vigorous at a temperature as high as 34° C. (Pl. XXII, fig. 2), its maximum no doubt lying just beyond 37° C. The optimal temperature seemingly lies over a fairly wide range, 27–32° probably being sufficiently inclusive. Infection can occur and progress, on the other hand, at a relatively low temperature, the minimum being below 15.° Since the incubation period for this disease is very short, it is not surprising that it occurs in the early spring and in northerly tobacco sections; but, on the other hand, it will probably never be as serious in the northern as in the southern districts.

The maximum temperature for the growth of *Bacterium tabacum* in culture apparently lies close to 35° C. The optimal temperature could not be definitely established, but from cultures run in the controlled chambers and in other incubation chambers it apparently lies between 25 and 30°. At any rate the "wild-fire" organism seemingly is capable of attacking the tobacco leaf at a temperature at which it will not grow in culture on potato agar. These maxima lie fairly close together and it may be that the temperature of a respiring leaf is somewhat below that in a culture tube set in the same chamber.

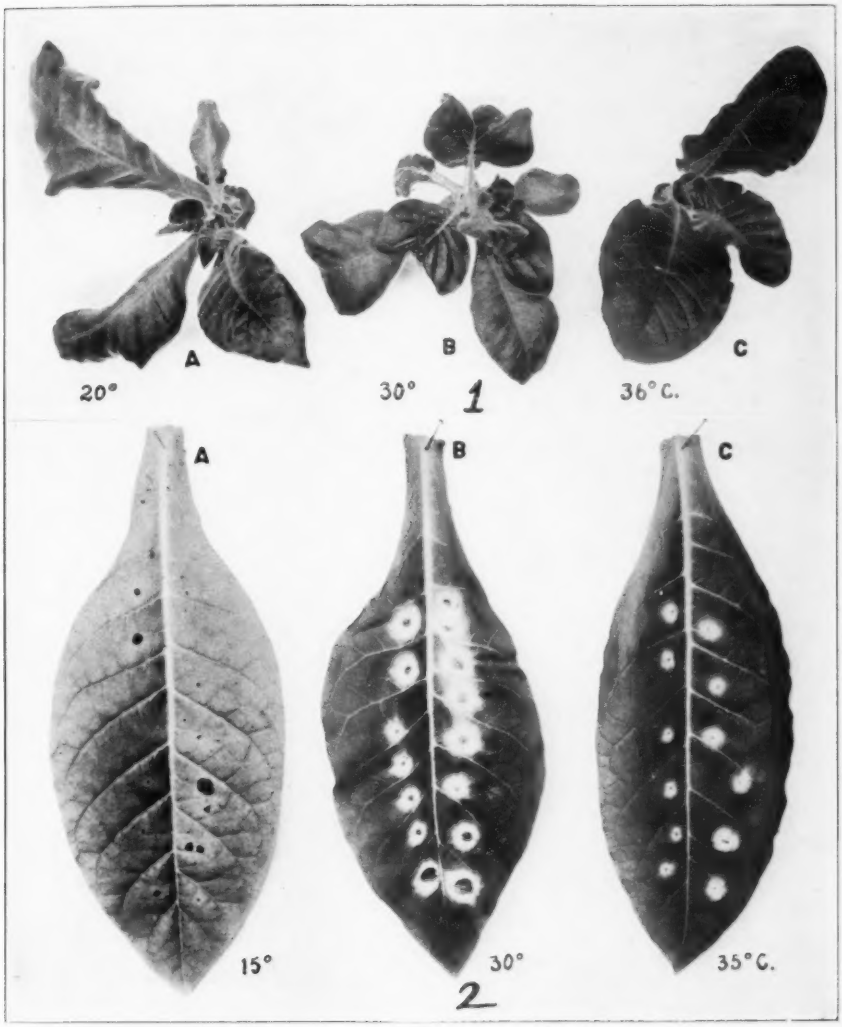
THE LATE BLIGHT OF THE POTATO

The influence of environment on the Phytophthora disease of potatoes is of especial interest on account of the large volume of observational data which has been published concerning this disease. The literature seems to be fairly well divided in considering cold humid, and warm humid weather most favorable for the occurrence of the disease. The work of Melhus (5) has shown the importance of chilling in determing the amount of infection, although the development of the disease is said to be favored by higher temperatures. The writer's work upon this disease has not as yet been sufficiently extensive to warrant a detailed discussion of this subject, but it is hoped that at a later date, adequate consideration may be given to it. In the experiments thus far conducted the plants were usually inoculated and allowed to remain outside the chambers until signs of infection occurred. They were then placed in the chambers at the different temperatures. Seven series of experiments were conducted but good infection was secured in only five of the series. The results indicate that the progress of the disease below 25° C. is insignificant as compared with that at higher temperatures. The optimum appears to be between 25 and 32° and maximum above 36° C. (Pl. XXIII, fig. 1.). The most important fact brought out in these tests in view of the opinions expressed in relatively recent literature is that Phytophthora is a relatively vigorous parasite at temperatures as high as 32–35° C. The action of the fungus on the host corresponds quite closely with its growth in culture in the same temperature chambers, although again the fungus appears to be relatively more active on the host plant than in the culture tube. According to Jones, Lutman and Giddings (4) growth did not occur on Lima bean agar at 30° C. In my tests appreciable growth was secured on potato agar at 35–36° C.

The results are believed sufficient at least to justify the conclusion that the late blight of potato is favored by relatively warm, humid weather rather than by cool, humid weather as far as the development of the disease is concerned, although original infection, at least by



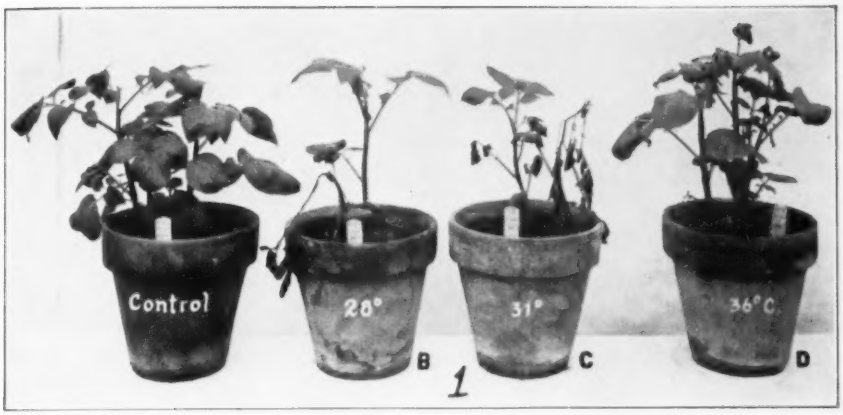
TEMPERATURE AND HUMIDITY REGULATING APPARATUS

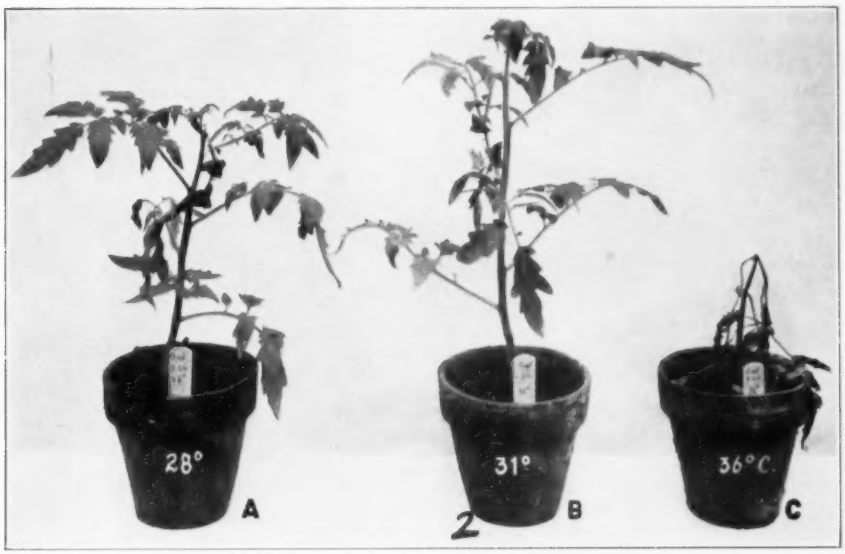


TOBACCO MOSAIC AND WILD FIRE GROWN IN CONTROLLED CULTURE CHAMBERS

FIG. 1. Illustrating the influence of air temperature on mosaic of tobacco. A. Inoculated and grown at 20° C. for 14 days. B. Inoculated and grown at 30° C. for 14 days. C. Inoculated and grown at 36° C. for 14 days. The plants were then exposed for an additional 14 days to ordinary greenhouse temperature (18–24° C.) with the plant exposed to 36° C. remaining free from mosaic symptoms.

FIG. 2. Showing the influence of air temperature on the "wild fire" disease of tobacco. A. Inoculated leaf from plant held at 15° C. B. Inoculated leaf from plant held at 30° C. C. Inoculated leaf from plant held at 35° C.





POTATO AND TOMATO WILT GROWN IN CONTROLLED CULTURE CHAMBERS

FIG. 1. The influence of air-temperature on the late blight of the potato. A. Inoculated with Phytophora and held under similar conditions to B, C, and D, until signs of infection occurred. A was then left under the ordinary greenhouse conditions and, B. Kept at 28° C. for four days. C. Kept at 31° C. for four days. D. Kept at 36° C. for four days.

FIG. 2. The influence of air temperature on the bacterial wilt of tomato (*B. solanacearum*). A. Inoculated and kept at 28° C. for five days. B. Inoculated and kept at 31° C. for five days. C. Inoculated and kept at 36° C. fou five days. Some infection may be noted in A and B.

zoospores is undoubtedly favored by low temperatures as shown by Melhus (5). Consequently late potato blight should be most severe according to these studies, when the above named conditions follow each other in the proper order and persist the proper interval of time.

SUMMARY

1. A series of chambers is briefly described in which the air-temperature and humidity are controlled within relatively narrow limits, while the growth of green plants is permitted.

2. A study has been made of the influence of air-temperature upon the mosaic disease of tobacco, a bacterial leaf-spot of tobacco (*Bacterium tabacum*) and upon the late blight of potato (*Phytophthora infestans*).

3. The optimal temperature for the development of mosaic lies between 28–30° C., and the maximal temperature at about 36–37° C. Mosaic plants placed at 36–37° send out new leaves, showing no symptoms of disease, and chlorotic leaves may recover their normal color. The temperature curve of this activity of the mosaic virus corresponds more closely to that of plant pathogenic organisms than to that of enzymatic or chemical action.

4. The activity of the enzymes, to which mosaic has been attributed, does not appear to be lowered at the maximal temperatures for the development of the disease and these enzymes are believed therefore to be an effect rather than a cause of the mosaic disease.

5. The optimal temperature for the "wildfire" disease of tobacco lies between 28–32°; the minimum is below 15; and the maximum above 37° C.

6. The optimal temperature for the development of the Phytophthora blight of potato lies above 25° and below about 32,° and probably quite close to the latter. The maximum lies close to 36–37° C. These results are believed to have some bearing upon the explanation of the occurrence and severity of the late potato blight disease.

THE WISCONSIN AGRICULTURAL EXPERIMENT STATION
AND THE
OFFICE OF TOBACCO INVESTIGATIONS,
UNITED STATES DEPARTMENT OF AGRICULTURE

LITERATURE CITED

(1) CHAPMAN, G. H. Mosaic disease of tobacco. Massachusetts Agric. Exp. Sta. Bull. 175: 73–117. *5 pl.* 1917.

(2) DOOLITTLE, S. P. Influence of temperature on the development of mosaic diseases. (Abstract) Phytopath. **11**: 46–47. 1921.

(3) HOTTES, C. F. A constant humidity case. (Abstract) Phytopath. **11**: 51. 1921.

(4) JONES, L. R., N. J. GIDDINGS AND B. F. LUTMAN. Investigations of the potato fungus Phytophthora infestans. United States Dept. Agric. Bur. Plant Indust. Bull. 245. *100 p., 10 pl. (pl. 1–2 col.).* 1912. Index to literature, p. 88–93.

(5) MELHUS, I. E. Germination and infection with the fungus of the late blight of potato. Wisconsin Agric. Exp. Sta. Res. Bull. 37. *64 p., 8 fig.* 1915. Literature cited, p 61–64.

(6) WOODS, A. F. Observations on the mosaic disease of tobacco. United States Dept. Agric. Bur. Plant Indust. Bull. 18: 1–24. *6 pl. (pl. 1, 2 and 6 col.)* 1902.

PLATE XXI. APPARATUS TO REGULATE TEMPERATURE AND HUMIDITY IN CULTURE CHAMBERS

FIG. 1.—General view of front of air-control chambers. The chamber to the right shows the brine pipes, the table on which most of the plants were grown, and the fan used for air circulation.

FIG. 2. Rear view of the air-control chambers. A. Motor used to operate the brine pump. B. Ice box in which the primary brine coils are cooled. C. Relay operating the brine pump motor through thermostat. D. Check to prevent flow of air from chamber to the outside of the greenhouse or vice versa through the blower (E). E. Blower for cooling by drawing air from outside the greenhouse during cold weather, supplementing the brine system. F. Motor operating the fans in the chambers. G. Thermostated hot-water tank for heating the spray. H. Drain from "spray-tanks."

FIG. 3. Humidifying apparatus. I. Blower used for forcing air through spray in tank (J) into chamber. J. "Spray-tank" in which the spray is confined. K. One of the two relays used in the "double-control" of temperature. L. Hot and cold water pipes running into "mixer."

FIG. 4.—G. Hot-water tank, heated with steam. P. Thermostat controlling the temperature of the water in the tank.

THE LIFE OF PUCCINIA MALVACEARUM MONT. WITHIN THE HOST PLANT AND ON ITS SURFACE

JAKOB ERIKSSON

Since the year 1912 I have made extensive investigations upon the life history of the hollyhock rust, *Puccinia malvacearum*. From the detailed report on the results of these studies, entitled "Das Leben des Malvenrostpilzes in und auf der Nährpflanze" which will be published soon (Kgl. Sv. Vet. Akad. Handl. Stockholm), I give some short extracts.

I. DISEASED AND SOUND HOLLYHOCK RACES

In the year 1913 I cultivated in the experiment garden at Experimentalfältet (Stockholm) two different hollyhock races of differing origin. All plants were grown from seeds in June, 1912. The one race grew from September 1 to October 30, severely affected by the rust. The other race kept quite sound during the same period. The plants hibernated out of doors, covered with leaves and hatches.

On May 2, 1913, 32 plants of the diseased race were planted in 8 zinc cylinders, 1.3 meters deep and 0.62 meters wide, open at the bottom, embedded in the ground and filled with fertile soil. On the same day 32 plants of the sound race were planted in 8 cement boxes, 1 meter square, also buried in the ground and filled with fertile soil. The distance between the two cultures was from 10 to 15 meters.

All vessels were watered from time to time. From June 3 to August 23—June 3, 9, 18, 27, July 4, 25, 29, 30, August 8, 23—every leaf of every plant was carefully examined. The degrees of rust were indicated as follows: 1 = 1 to 10 sori, 2 = 10 to 25, 3 = 25 to 100 and 4 = more than 100 sori.

In table 1, I have assembled the results from 2 cylinders (8 plants) of the affected race and from 2 boxes (8 plants) of the sound race.

From this table we can learn two things: 1. There are two different periods of disease, caused by this fungus: the first period from the beginning of May to the end of July and the second from the end of July to the beginning of cold weather. During the first period the attack is slight, but during the second very bad. 2. The sound race of plants, in spite of the short distance between the two cultures, grew quite clean up to the last week of July. In 2 or 3 days (July 26 to 28) suddenly there appeared as at one blow an extreme outbreak of rust sori on all full grown leaves of all plants on sound as well as on affected races.

In the several years the sanitary state of hollyhock races, which had been experimented with, changed essentially. This may be seen in table 2.

According to this table, I worked during the years 1912–1916 with 7 races which I characterize as "affected originally and finally," and with 2 races (1913 and 1914), characterized as "originally sound, but during the spring and the summer of the second year infected from the diseased plants of the other race," growing close to them. In the years 1917 to 1920 I worked with 4 races sound from the beginning to the end of the culture, with 1 (or 2) races (1919), affected the whole time, and with 2 races (1918 and 1920), which changed from affected to sound.

TABLE 1

Hollyhock rust at Experimentalfältet (Stockholm) in the year 1913

DAYS	RACE AFFECTED 2 CYLINDERS—8 PLANTS					LEAVES DEAD	TOTAL	RACE SOUND 2 BOXES—8 PLANTS					LEAVES DEAD	TOTAL
	NUMBER OF LEAVES WITH DEGREE OF RUST							NUMBER OF LEAVES WITH DEGREE OF RUST						
	0	1	2	3	4			0	1	2	3	4		
3-6	61	1	1				63	All leaves clean						
9-6	45	15	2	1			63	"	"	"				
18-6	30	8	14	8	1	2	63	"	"	"				
27-6	21	6	8	21	2	5	63	"	"	"				
4-7	21	5	3	21	6	7	63	"	"	"				
25-7	12	16	8	24	12	33	105	"	"	"				
29-7	13	18	13	17	54	36	151							
30-7								28	15	13	18	61		135
8-8								30	15	13	15	61		134
23-8	35	22	27	10		74	169	13	11	14	25	69	36	168

Further, it is to be remarked that the summer stage of the disease quite or partially failed to appear in the years 1915 to 1920. Nevertheless the disease appeared in the autumn stage in the years 1915, 1916 and 1919 on the young plants of the first year as well as on the old plants of the second year.

II. WATERING OF THE CULTURE SOIL WITH FUNGICIDES

In the year 1913, I also made some experiments of watering certain plants with a diluted solution of copper sulphate. The results of these experiments are to be found in table 3.

From this table, we can conclude that copper sulphate is an effective means to stop the life energy of the fungus during the summer period of the disease, from the earliest spring to the last week of July, but

that it is without any effect whatever during the autumn period of disease, from the end of July to the beginning of winter. The most favorable concentration of the copper solution seems to be 0.3 per cent.

Upon the sound race of plants no effect of the watering with the fungicide was to be detected either in the first or in the second period

TABLE 2

Sanitary state of hollyhock races in 1912–1920

(Bergianum = Bergian Garden (Stockholm); Haga = Haga Garden (Stockholm); Hilleshög (Skåne); Västeras (Västmanland).

YEARS	ORIGIN	FIRST YEAR	SECOND YEAR			RACE LIKELY	
		AUTUMNAL OUT-BREAK (PROLEPTIC)	CLEAN PERRIOD	FIRST RUST PERIOD (SUMMER STAGE)	SECOND RUST PEIOD (AUTUMNAL STAGE)	ORIGINALLY	FINALLY
1912	Haga	bad 15/8–15/9	3/5–18/6	bad 26/6–15/7	bad 31/7–17/8	affected	affected
1913	Haga	bad 1/9–30/10	2/5–24/5	bad 24/5–4/7	bad 25/7–23/8	affected	affected
	Bergianum	O	3/6–25/7	O	bad 28/7–22/8	sound	affected
1914	Haga	bad	O	bad 1/5–25/7	bad 27/7–	affected	affected
	Bergianum	O	2/5–8/5	slight 9/5–25/7	bad 27/7–	sound	affected
1915	Hilleshög	slight 3/10–10/11	20/5–10/7	O	bad 16/7–6/9	affected	affected
	Haga	very slight 15/10–3/11	20/5–10/7	O	bad 16/7–6/9	affected	affected
1916	Bergianum	very bad 9/9–3/10	Ser. A 17/5–12/7	O	bad 19/7–9/8	affected	affected
	Bergianum	very bad 9/9–3/10	Ser. B-D O	slight 17/5–12/7	bad 19/7–9/8	affected	affected
1917	Bergianum	?	24/5–23/8	O	O	sound	sound
1918	Bergianum	very bad 2/11	6/5–20/8	O	O	affected	sound
	Västeras	?	23/5–15/9	O	slight 4/8–15/9		affected
1919	Haga	very slight	19/5–15/9	O	bad 4/8–15/9	affected	affected
	Bergianum	O	19/5–15/9	O	O	sound	sound
	Bergianum	very bad 2/19–18/10	15/5–23/8	O	O	affected	sound
1920	Västeras	O(?)	15/5–23/8	O	O	sound	sound
	Haga	O(?)	15/5–23/8	O	O	sound	sound

of the disease. The great difference in the effects of the watering with the fungicide in the different periods of disease shows evidently that this fungus must live in the tissues of the host plant in another mode of existence during the spring and summer (May–July) than during the autumn (August–October).

The fact that a fungicide added to the water used for watering the culture soil can restrain the fungus element in the symbiose, without damaging the normal element of the host cell itself, must be regarded as the introduction of a new method of combatting the diseases of plants, i. e., a method of internal therapeutics.

TABLE 3

Watering experiments in the year 1913, each series is represented by 8 plants in 2 cylinders.

DAYS	SER. A: WATER							SER. B: 1% COPPER SULPHATE						
	NUMBER OF LEAVES WITH DEGREE OF RUST					LEAVES DEAD	TOTAL	NUMBER OF LEAVES WITH DEGREE OF RUST					LEAVES DEAD	TOTAL
	0	1	2	3	4			0	1	2	3	4		
June 3	61	1	1				63	55	8					63
" 9	45	15	2	1			63	46	13	2	2			63
" 18	30	8	14	8	1	2	63	38	17	4	2		2	63
" 27	21	6	8	21	2	5	63	37	15	4	2		5	63
July 4	21	5	3	21	6	7	63	43	7				13	63
25	12	16	8	24	12	33	105	44	9				30	83
29	13	18	13	17	54	36	151	19	16	14	16	36	32	133
Aug. 23	35	22	27	10		74	168	49	26	22	6		62	165

DAYS	SER. C: 0.3% COPPER SULPHATE							SER. D: 0.2% COPPER SULPHATE						
	NUMBER OF LEAVES WITH DEGREE OF RUST					LEAVES DEAD	TOTAL	NUMBER OF LEAVES WITH DEGREE OF RUST					LEAVES DEAD	TOTAL
	0	1	2	3	4			0	1	2	3	4		
June 3	59	3					62	63	3					66
" 9	48	13	1				62	45	18	2	2			67
" 18	47	10	2			3	62	49	12	3	1		3	68
" 27	45	12	1			7	65	45	9	2	1		12	69
July 4	54	4				7	65	48	5				16	69
" 25	53	26				21	100	52	26	3			35	116
" 29	33	29	18	19	13	25	137	36	24	15	24	25	44	168
Aug. 23	60	16	19	7		13	155	58	13	15	19		69	174

III. GERMINATION OF THE SPORES

All spores of this fungus are two-celled and morphologically equal, but in reality we have to distinguish between two biologically different forms of these spores, different in the manner of germination as well as in their method of producing infection.

The one spore form is that of the autumn spores, which occur on the young plants of the first year and on the old plants of the second year, in the months of August until October. These spores are able to germinate in two different manners. If these spores lie on the surface of a drop of water or in moist air they germinate with short and curved basidia (promycelia), which are generally divided into 4 segments. From the side of each one of these segments a basidiospore (sporidium) is separated. If these autumn spores lie submerged in water they germinate with long and straight tubes, which at the tip liberate bud-cells (conidia), arranged like a string of beads. The other form of spores is the summer spores, which occur on the hibernated plants of the second year, in the months of May until July. These spores whether lying upon or under water germinate only in one manner, with long and straight tubes which liberate conidia.

In some cases the mycelium of the autumn generation survives in the tissues of the host plant from the first year to the second year and during the spring and the summer continues in developing autumn spores which germinate in the two ways above mentioned, short as well as long. That occurred in my experiments (Bergianum) in the year 1914, and to a certain extent also in the year 1916. In other cases the summer spores failed to appear at all. That occurred in my cultures (Bergianum) in the years 1915, 1917, 1919 and 1920. In yet other cases only the summer-spore stage is developed and this stage kills all the leaves of the plants already in July. That occurred (Malmô, South Sweden) in the year 1920 on old hollyhock plants and on new plants of *Malva silvestris*.

The two spore-forms are different also in another regard: on reaching a leaf of a plant susceptible to the fungus, the sporidium penetrates the epidermis by means of a germ tube and produces after 8 to 10 days new rust sori on its surface. On the contrary on reaching such a leaf the conidium infuses its contents as plasm in the epidermal cell, the nucleus of this cell becomes hypertrophied and the plasm penetrates into the interior of the leaf to constitute a mycoplasm. In this case no new sori are developed on the infected leaf; only sharp, limited, dead spots, finally white, or none at all, appear on those places, where the inoculum had been placed.

By proving that there are two different forms of spores, different in regard to their germination and to their infecting power, the surprising phenomena of the year 1913—a race of hollyhocks sound in the months of May until the end of July, growing side by side with an affected race—have now cleared up, as well as many heretofore inexplicable, often contradictory statements of observations in the older as well as in the modern professional literature.

STOCKHOLM.

EUROPEAN CANKER ON THE PACIFIC SLOPE

S. M. Zeller and C. E. Owens

With Four Figures in the Text

Since January, 1918, when Professor H. P. Barss identified the European canker on the Red Cheek Pippin apple in Marion County, Oregon, specimens of this trouble have been received by the Department of Botany and Plant Pathology, of Oregon Agricultural College, from various localities in Oregon and one in California. It has been reported from four points in Marion County, one in Hood River, one in Clatsop, one in Tillamook, eight in Benton, one in Linn, one in Lane and one in Douglas County. Other suspicious cankers have been observed in Hood River and Wasco Counties but since no fruiting stages were in evidence positive identifications were not made at the time of collection. In April, 1920, we received a specimen of European canker on apple from Humboldt County, California, sent in by Professor Elizabeth H. Smith, University of California.

Among the specimens which have been identified as due to *Nectria galligena* Bres. were several cankers on apple the varieties of which were unknown to us. The hosts for this canker upon which the perfect stage (*Nectria galligena*) or the imperfect stage (*Fusarium Willkommi* Lindau) have been definitely determined for the region under consideration are several apple varieties, namely, Red Cheek Pippin, Bismark, Delicious, Winter Bellflower, Spitzenburg, and Newtown and also D'Anjou, Howell and Bosc pears.

In Europe and the northeastern part of the United States the European canker on apple is described as a slow-growing disease which enters the host through some wound or bruise, usually where a small twig or spur has been broken or pruned off, where the strain on crotches has caused a crack, and through such bruises as may occur by the impact of hailstones upon the bark. It spreads slowly, killing the bark as it goes. Each year the bark at the periphery of the canker is encroached upon by the fungus and is killed, thus causing a new callus to form just outside of the last one. As the disease thus spreads over a period of years a number of concentric calluses are formed. The dead bark falls away exposing the wood so that these concentric wood calluses show up conspicuously, giving the entire canker its striking appearance, which is characteristic

of the open type of canker as it occurs there. This type of canker is reported as prevailing especially on erect, vigorous scaffold branches of the tree while a closed, rough gall-like canker is characteristic of the horizontal or hanging branches.

However, on the Pacific Slope the concentric arrangement of callus rings in old cankers is much less in evidence and usually can not be found in most cankers caused by *Nectria galligena*. For some reason, possibly due to climatic or other conditions not yet fully understood, the cankers spread very rapidly, sometimes extending for several inches or even one

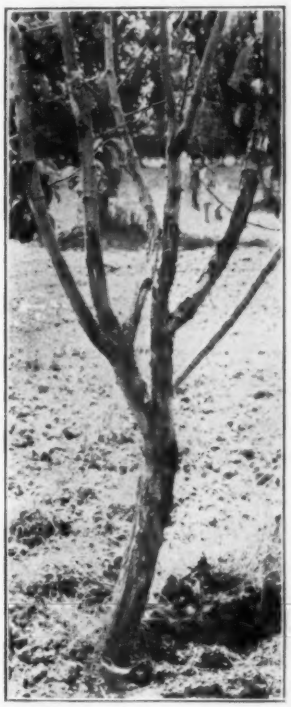


FIG. 1. A CROTCH INFECTION ON D'ANJOU PEAR. THE CANKER HAS FOLLOWED WINTER INJURY IN THIS INSTANCE AND IS IN ITS SECOND YEAR.

or two feet in a single season and remain more or less closed. One can readily imagine that this continual lateral and longitudinal growth without callus formation can be possible only in a locality where there is a prolonged humid season with moderate temperature such as usually prevails for several months on the Pacific Slope of the Northwest. This perhaps is the reason that we find in western Oregon these large cankers covering many square inches of bark surface without the conspicuous formation of calluses.

For the most part there is every indication that infections take place at small, sappy, pruning cuts or in crotches. On the Red Cheek Pippin apple and Bosc and D'Anjou pear, cankers have been observed frequently to start in the crotches of the largest branches, as shown in figure 1. In many instances where there are no pruning cuts there are indications of infection in the crotches of fruiting and leaf spurs. Wherever the bark

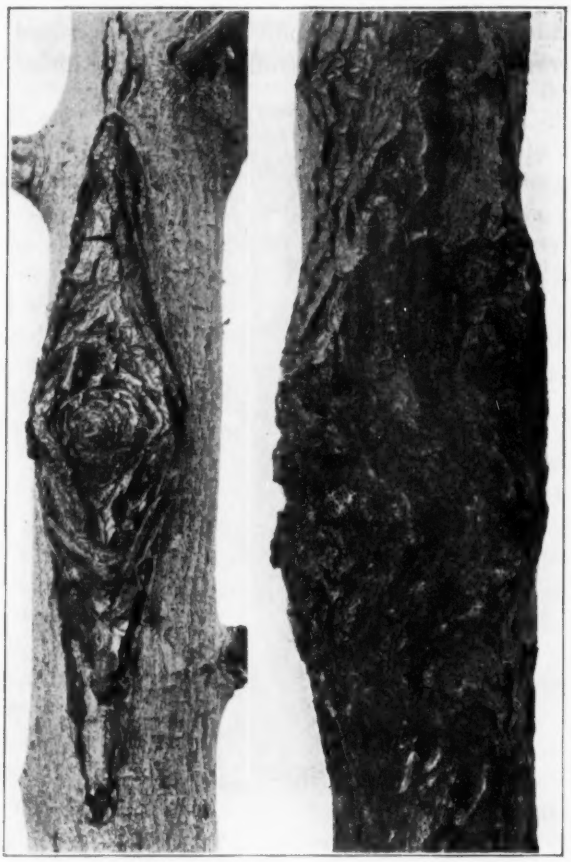


FIG. 2 FIG. 3

FIG. 2. A TYPICAL ONE-YEAR-OLD CANKER ON BOSC PEAR. INFECTION STARTED AT A SMALL DEAD SPUR. NOTICE THE ROUGH, CLOSED TYPE OF CANKER

FIG. 3. A CLOSED CANKER ON THE RED CHEEK PIPPIN. THIS CANKER IS SEVERAL YEARS OLD

of the host has become devitalized, through sun scald or winter injury, for instance, the fungus obtains an easy access and from there spreads to the healthier tissues. In some cases it has been found that the fungus has obtained its start in the periphery of cankers produced by *Neofabraea*

malicorticis on D'Anjou pear. In an orchard of Bosc pears in Benton County where nearly every tree has some cankers of *Nectia galligena* there is considerable bark injury due to the freeze of December, 1919. From many of these winter-injured areas cankers produced by *Nectria galligena* are spreading with extreme vigor. In most cases of such winter injury both to Bosc and D'Anjou pear, *Nectria cinnabarina* (Tode) Fr. has also gained a foot-hold and there is much evidence that this fungus although usually saprophytic, exists as a parasite at least upon partially devitalized bark and wood.

Although the life history of *Nectria galligena* should be traced definitely for the humid conditions under which it is found here there are some observations which have already been made which we believe should be presented at this time. Near Corvallis the sappy, soft cankers upon which the macro- and microconidia occur have been observed on D'Anjou pear during the early part of June. For the most part these cankers occur around early-spring pruning cuts and unmistakably are due to infections of this spring. By the middle of June many of these cankers had already reached 20–22 inches in length and 1.5–2.5 inches in breadth. If the bark around such cankers is removed it will be found that in most cases the fungus has destroyed the cambium far beyond the superficial limits of the cankers. If the bark is cut through at the margin of the canker where the cambium is thus destroyed the bark will shrink away from the wood exposing a slimy wood surface, the limits of which are definite, making it possible to cut off the bark beyond the limits of this cambium infection, when disinfection and antiseptic methods are applied.

In contrast to the above-mentioned advance of the fungus in the cambium there seems to be a very different type of canker formed at times on pear bark, perhaps by another organism, in which there is no apparent damage to the bark deeper than the inner cortex. In these cases the cambium is apparently in perfect health and remains in this condition during the first season of the life of the canker. This type of canker has all the appearance of the early stages of European canker but we have never seen fruiting bodies of *Nectria galligena* in connection with it.

Upon cankers caused by *N. galligena* and which we suspect were formed the same season the senior author has found pustules of the multi-septate, sickle-shaped macroconidia as well as the ellipsoid, one- and two-celled microconidia. These were found on apple cankers collected February 3, 1920 and on pear cankers collected October 13 of the same year. The white stromatic cushions bearing these conidia are shown in figure 4. The microconidia and macroconidia to date have been found in

early spring and lasting at least until October. The stromatic cushions of conidia first appear very near the seat of infection and later break out as pustules further out from this point as the canker increases in size. Embedded in these stromatic pustules of conidiophores are to be found the early stages of perithecia the development of which will be described later by one of us.

In the very early stages of the canker, especially on pear, the bark becomes undulating, the raised portions having a spongy edematous character. Following this edematous condition the epidermis peels off irregularly giving a more or less shaggy appearance to the new oozy canker. The unbroken epidermis easily slips off upon pressure with the finger. The exposed portions of the cortex are then black and moist.

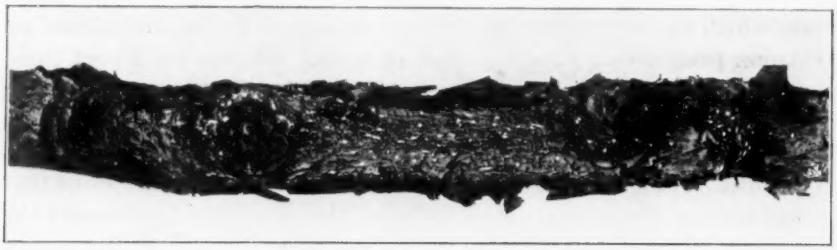


FIG. 4. CANKER ON D'ANJOU PEAR SHOWING THE WHITE STROMATIC PUSTULES OF THE FUSARIUM STAGE OF THE FUNGUS, NECTRIA GALLIGENA

As the cankers become older the killed bark becomes very much cracked and furrowed, sometimes along irregular lines, but many times especially where the disease made its first vigorous onslaught these cracks take on a more or less concentric arrangement although of course these concentric fissures do not represent annual growth periods as in the case with the open type of canker found in other localities, but may represent merely a periodicity of growth of the fungus due to changes in moisture or temperature during a single season. This is perhaps similar to the closed type of canker reported from other regions, but evidently is much more rapid in growth. Occasionally in this state cankers are found upon apple which for some reason (possibly host resistance) have developed more slowly and in such cases there may be a slight tendency toward an open concentric callus formation.

OREGON EXPERIMENT STATION,
CORVALLIS, ORE.

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